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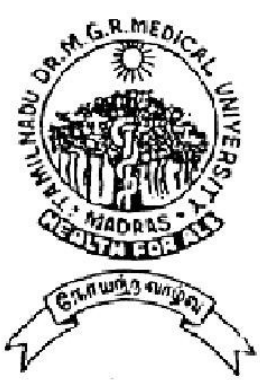

STUDY OF 50 CASES OF MALIGNANT OBSTRUCTIVE JAUNDICE

DISSERTATION SUBMITTED FOR

BRANCH – I M.S. (GENERAL SURGERY)

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DEPARTMENT OF GENERAL SURGERY

GOVT. RAJAJI HOSPITAL

MADURAI

CERTIFICATE

This is to certify that the dissertation entitled **"STUDY OF 50 CASES OF MALIGNANT OBSTRUCTIVE JAUNDICE"** submitted by **Dr. K. SENDHIL NATHAN** to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.S Degree Branch – I (General Surgery) is a bonafide research work and was carried out by him under direct supervision & guidance.

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DECLARATION

I, **Dr. SENDHIL NATHAN. K,** hereby declare that I carried out this work on, **"STUDY OF 50 CASES OF MALIGNANT OBSTRUCTIVE JAUNDICE"** at the Department of Surgery, Govt. Rajaji Hospital, Madurai during the period of June 2006 to November 2008. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree or diploma to any other University, Board either in India or abroad.

This is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.S degree examination in General Surgery.

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CHAPTER I

INTRODUCTION

Surgery as the modality of treatment for jaundice is not fully acceptable to a large majority of population in our part of the country. May be because of high belief in ayurvedic medicine, which is accepted as the best remedy for jaundice, and probably due to lower incidence of obstructive jaundice in our population in the past. Anyhow there is an increasing evidence of obstructive jaundice especially malignant obstructive jaundice. Surgeons thus face an increasing number of patients with obstructive jaundice reaching them in a fairly advance stage.

The fundamental problem met with in dealing with a patient with prolonged jaundice is the accurate diagnosis of its cause whether obstructive or not and if obstructive what exactly its cause.

In managing malignant obstructive jaundice the problem of diagnosis becomes an acute one because jaundice caused by mechanical obstruction to common bile duct should be surgically remedied whereas in the absence of mechanical block of bile duct treatment becomes medical. The accurate diagnosis of mechanical obstruction to CBD becomes difficult at times because the clinical features and biochemical investigation may be atypical. Intrahepatic cholestasis gives rise to clinical features and laboratory data similar to mechanical block of common bile duct. Many times hepatocellular damage and mechanical obstruction coexist making the diagnosis much more difficult.

Treatment of malignant obstructive jaundice is challenging. Surgical treatment ranges from definitive surgical procedures to palliative procedures. Non operative management includes endoscopic stenting, and interventional radiological procedure like PTBD. All these tests the surgeon because of relative inaccessibility of the extrahepatic biliary tree and pancreas.

CHAPTER II

REVIEW OF LITERATURE – I

ANATOMY OF BILIARY TREE

The biliary tract begins as a series of blind ending channels or canaliculi formed by specialized structures in the cell membrane of adjoining hepatocytes. These canaliculi drain into the ductules which initially are single layered epithelial tubes and later a muscular tube with inner epithelial lining. These ductules join together to form the interlobar duct emptying in the right and left hepatic ducts emerging from each lobe of the liver. These hepatic ducts unite to form the common hepatic duct, which receives the cystic duct to become the common bile duct. The common bile ducts in most of the cases before entering the duodenum receives the pancreatic duct and the two share a final channel for about 7 mm in length within the pancreas. Thus the biliary tree has

1. Hepatic component
2. Extrahepatic component
3. Pancreatic component

Defects in the form of blockade anywhere in the hepatobiliary pancreatic system thus results in cholestatic jaundice.

GROSS ANATOMY

LIVER

Liver is the largest gland in the body. It is almost completely covered with peritoneum, which is reflected on to the adjacent structures forming the so called falciform ligament of the liver. The region devoid of peritoneum is the bare area of the liver.

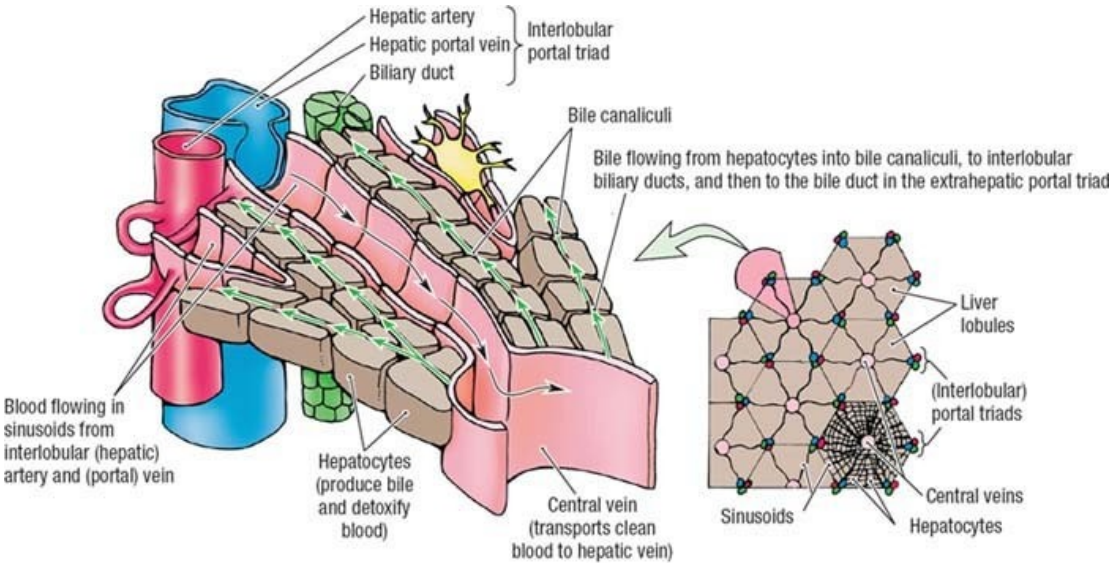
The liver has got diaphragmatic and visceral surfaces. The diaphragmatic surface has anterior, posterior, superior and lateral surface or parts. The bare area of liver comes in the posterior surface of the liver. The porta hepatis and the gall bladder are related to the visceral or inferior surface.

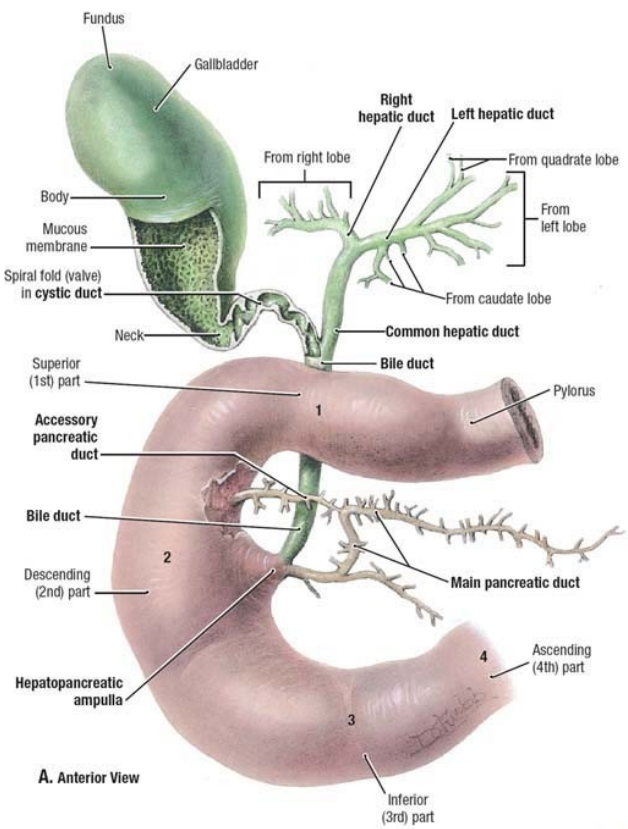
Grossly the liver is divided into a larger right and a smaller left lobe by the attachment of the falciform ligament on the superior and anterior surface of the liver and the groove for ligamentum venosum on the posterior surface. Thus the anatomical right lobe includes the quadrate lobe on the inferior surface and caudate lobe on the posterior surface.

From the surgical point of view the more important one is the functional right and left lobes of the liver, the dividing line pass through the gall bladder bed anteriorly and inferiorly and through the groove for the inferior vena cava posteriorly. This functional division is based on the territory of arterial supply, venous and biliary drainage. Thus the caudate and quadrate lobes belong to the functional left lobe of the liver.

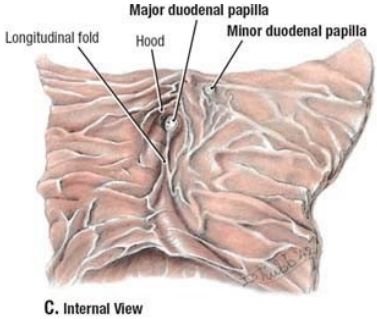
HEPATIC DUCTS

One hepatic duct each from the functional right and left lobes of the liver emerge and join together about 1-2 cms. below the porta hepatis to form the common hepatic duct. The left hepatic duct is always a single channel but on the right side, two separate ducts may exist in about 25% of cases, which join the left hepatic duct independently.

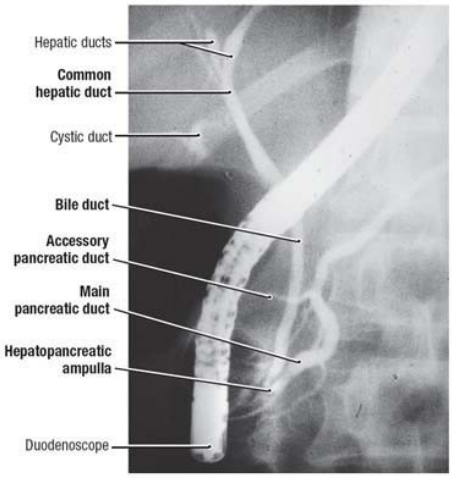




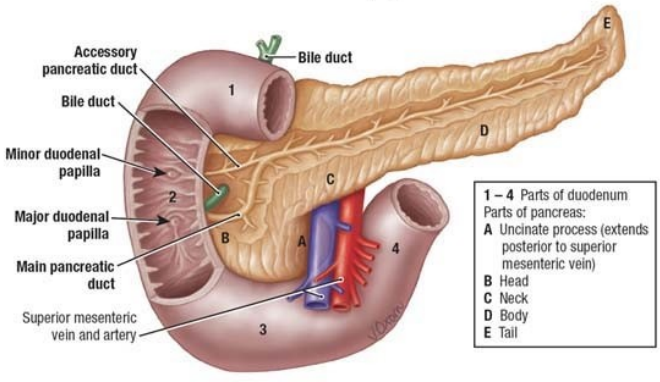
A. Anterior View



C. Internal View



D. Anterior View



1 – 4 Parts of duodenum
Parts of pancreas:
A Uncinate process (extends posterior to superior mesenteric vein)
B Head
C Neck
D Body
E Tail

B. Anterior View

COMMON HEPATIC DUCT

It is formed by the union of the two hepatic ducts and measure about 2-4 cms in length with an average inner diameter of 8 mm. It lies in the free border of the lesser omentum to the right and front of portal vein, with hepatic artery lying on its left side. The right branch of hepatic artery passes behind the common hepatic duct.

GALL BLADDER

The gall bladder is a pear shaped sac of about 7 cms in length with a capacity of about 30-50 ml. which lies in the gall bladder fossa in the under surface of the liver. It has a fundus, body, infundibulum and a neck. The gall bladder is covered with peritoneum on all sides except at the place where it is in direct contact with the liver. The wall of the neck where it becomes the cystic duct may show a small diverticulum directed downwards and backwards. This pouch is called the Hartmann's Pouch.

CYSTIC DUCT

It is the continuation of the neck of the gall bladder, and is about 4 cms long with an inner diameter of 2-3 mm. It passes up and to the left to join the common hepatic duct about 1-2 cm above the duodenum. The cystic artery commonly lies behind the cystic duct, it has spirally placed mucosal folds called Valve of Heister.

COMMON BILE DUCT

It measures about 7-8 cm long and consists of 4 parts. They are:

1. Supraduodenal	2.5 cm
2. Retroduodenal	2.5 cm
3. Paraduodenal/Intrahepatic	2.5 cm

4. Intraduodenal Very small

PANCREAS

Pancreas is a retroperitoneal elongated gland lying between the C loop of duodenum and splenic hilum. It has a head, body and tail with a small constricted part between head and body called the neck and another small downward projection from the head called the Uncinate Process. The main ductal system of the pancreas, the Duct of Wirsung, starts from the tail lying near the posterior than the anterior surface with small ducts of the lobes draining to it at right angles forming a herring bone pattern. It traverses the body and on reaching the neck it binds down posteriorly to join with the common bile duct to form a common dilated hepatico pancreatic Ampulla of Vater surrounded by the Sphincter of Oddi which prevents the reflux of bile into the pancreas and vice versa.

LYMPHATIC DRAINAGE

LIVER

Lymphatics from the liver go to different nodes. They are:

1. Hepatic nodes seen in the lesser omentum along the hepatic artery and bile duct.
2. Nodes along the inferior vena cava above and below the diaphragm.

GALL BLADDER

1. directly to the liver
2. to the hepatic nodes in lesser omentum
3. from the left side of gall baldder to the node in the Calot's Triangle.

EXTRA HEPATIC BILIARY TREE

From this region the lymphatic drainage is to hepatic nodes along the lesser omentum and to the node in the Calot's Triangle.

NERVE SUPPLY

The hepatobiliary tree gets nerve fibers from both parasympathetic (motor) and sympathetic (sensory) through vagus.

PHYSIOLOGY OF EXTRAHEPATIC BILIARY TREE

The primary function of extrahepatic biliary tree is to transport bile from the liver to the duodenum. Secondary function is to concentrate and modify the composition of bile.

TRANSPORT OF BILE

In animals with gall bladder, an organ which concentrates and stores bile, the flow of bile into duodenum does not parallel its secretion by liver. Between meals the ampullary sphincter offers certain degree of resistance to bile flow and hepatic bile enters the relaxed gall bladder and rest leaks into the duodenum.

The secretion pressure of bile in normal individual is 125 – 250 mm of water. This pressure is responsible for bile flow. The pressure in the biliary tree should be lower than the hepatic secretary pressure for maintaining the bile flow and it is usually 100-150 mm of water. The difference in pressure within the biliary passage and liver can be considered to be due to

1. The activity of Sphincter of Oddi
2. The filling of gall bladder
3. The resorptive activity of gall bladder

PHYSIOLOGY OF SPHINCTER OF ODDI

The sphincter maintains a brisk peristaltic motion and basic sphincter rhythm consists of opening and closing. The upper segment relaxes and opens and the ampulla gets filled up. The lower part of the sphincter then opens and bile flows into duodenum.

Studies, which recorded sphincter of Oddi functions by indirect methods, such as observing the rate of inflow of fluid into the common bile duct, concluded that the sphincter of Oddi remains closed during fasting, thus diverting bile flow into the gall bladder. After a meal the sphincter is relaxed and bile flow from the common bile duct into the duodenum is propelled by pressure generated by the gall bladder.

FILLING AND EMPTYING OF GALL BLADDER

Filling of gall bladder occurs when it is empty and its intraluminal pressure drops to 11-14cm of water. Evacuation of gall bladder occurs when the gall bladder musculature contracts and intra luminal pressure mounts upto 200-300 mm of water.

These functions are partly under the parasympathetic nervous control and largely under hormonal control by CCK.

RESORPTIVE ACTIVITY OF GALL BLADDER

The gall bladder mucosa reabsorbs the water and electrolytes entering it leaving behind bile salts, bile pigments and cholesterol. The active transport of sodium chloride and bicarbonate and obligatory water transport from bile to blood across the gall bladder mucosa results in concentration of bile.

CHAPTER III

REVIEW OF LITERATURE – II

JAUNDICE

The term 'Jaundice' is derived from the French word meaning 'Yellow' and refers to the presence of an excess of bile pigments in the tissues and the serum. It is a presenting sign of a number of hepatic and non-hepatic diseases. The differential diagnosis and management are dependent upon a appreciation of normal and abnormal variants of bile pigment metabolism.

PATHOPHYSIOLOGICAL CLASSIFICATION OF JAUNDICE

I. PREDOMINANTLY UNCONJUGATED HYPERBILIRUBINEMIA

A. Excess production of bilirubin

1. Hemolytic anaemia
2. Resorption of blood from large internal hemorrhages
3. Ineffective erythropoiesis

B. Reduced hepatic uptake

1. Drug induced
2. Prolonged fasting
3. Sepsis

C. Impaired bilirubin conjugation

1. Gilbert's Syndrome
2. Criggler-Najjar Syndrome I & II
3. Physiological jaundice of new born

4. Diffuse hepatocellular disease (hepatitis, cirrhosis)

II. PREDOMINANTLY CONJUGATED HYPERBILIRUBINEMIA (CHOLESTATIC JAUNDICE)

A. Decreased intrahepatic excretion of bilirubin

1. Dubin Johnson Syndrome
2. Rotor's Syndrome
3. Drug induced
4. Hepatocellular disease (viral hepatitis)
5. Primary biliary cirrhosis
6. Sclerosing Cholangitis

B. Extrahepatic biliary obstruction

1. CBD stones
2. Carcinoma of the head of pancreas, extrahepatic hile ducts and ampulla of Vater
3. Extrahepatic biliary atresia

NORMAL BILE PIGMENT METABOLISM

The bile pigment – bilirubin is a tetra pyrrole, which is formed to the greatest extent from hemoglobin and to a lesser extent from myoglobin breakdown and hepatic synthesis itself. When the red blood cell is destroyed by the reticuloendothelial system the iron and globin are removed and the heme ring is opened and transformed into biliverdin, which is green. The later is reduced to become bilirubin, which is yellow. The bilirubin combines with albumin to form a relatively stable protein-pigment complex and is transported as such to the hepatic parenchymal cell. This complex which is referred to as indirect reacting bilirubin, since it

gives the Vanderbergh diazo reaction only after treatment with alcohol and other substance that split the protein, is poorly soluble in water and is not excreted in the urine.

In the hepatic parenchymal cell the albumin is removed and the bilirubin is conjugated with glucuronic acid to form diglucuronide, which is water soluble and is excreted into the biliary canaliculi. This substance gives an immediate diazo reaction and hence termed as direct-reacting. This is passed into urine. Normally there is less than 1.2 mg of direct reacting serum bilirubin and less than 0.3 mg of indirect reacting bilirubin per 100 ml of serum.

The conjugated bilirubin which is excreted via the bile into the intestine is acted upon by bacteria and undergoes a series of reduction leading to the formation of two groups of compounds namely the colourless urobilinogen and coloured Stercobilin. The normal daily fecal excretion ranges between 40 and 300 mg with an average of 100-200 mg, and in newborn infants because of the absence of bacterial flora urobilinogen may be absent. A reduction in the enteric bacteria is also responsible for the reduced pigment excretion that accompanies the use of intestinal antibiotics. Some of the urobilinogen is reabsorbed by way of portal venous system and returns to the liver, where it is either removed or to a small extent excreted in urine.

ABNORMAL BILE PIGMENT METABOLISM

No classification is totally satisfactory. The classification most widely used distinguishes between hemolytic, obstructive and hepatocellular jaundice. However, it is most reasonable to categorize as

1. Those disease states in which the bile flow is unimpeded.
2. Those types that are associated with an impairment of the bile flow.

NORMAL BILE EXCRETION

The overproduction of bile pigment from excessive hemolysis creates a situation in which normal liver is confronted with more pigment than it is able to remove. This occurs in physiological jaundice of infancy and all pathological hemolytic states. However the reserve capacity of the liver is great and even when the bilirubin production is increased six times there is only a 2-3 mg rise in the serum bilirubin level per dL of serum. In this situation the increase in serum bilirubin is in the unocnjugated indirect reacting bilirubin, no bilirubin appears in the urine but there is an increase in the fecal and urinary urobilinogen. An excess of bilirubin production also occurs in shunt hyperbilirubinemia in which indirect bilirubin accumulates in the absence of any reduction in red cell life span.

Constitutional defects of liver function may also cause hyperbilirubinemia without impairment of bile flow. In Gilbert's disease there is defect in the bilirubin transport into the liver cell, while in Criggler-Najjar syndrome the defect is an inability of liver to conjugate the bilirubin with glucuronic acid. In these states, the elevation of bile pigments is in the indirect reacting fraction. All other hepatic function tests are normal, and no histological abnormality is noted. With all of the above mentioned diseases the bilirubin pigment is attached to albumin and cannot be excreted by the kidney, thus prompting the term acholuric jaundice.

IMPAIRED BILE EXCRETION

All other diseases are associated with an accumulation of conjugated bilirubin in the blood and impaired excretion. The bilirubin pigment which is water soluble, is readily excreted into the urine, which becomes brown. The obstruction may be intrahepatic or extrahepatic.

INTRAHEPATIC OBSTRUCTIVE JAUNDICE

In the Dubin-Johnson Syndrome, which is associated with the appearance of iron free pigment in the hepatic cells and normal liver function, the hepatic excretion of conjugated bilirubin is impaired. Intrahepatic cholestasis has also been related to a variety of drugs and hepatocellular disease. Methyltestosterone and norethiandrolene damage the microvilli of the bile canaliculi and may cause jaundice. The phenothiazine drugs such as chlorpromazine may evoke a hypersensitivity reaction in a small percentage of patients and result in cholangitic hepatitis and intrahepatic cholestasis. A lesion along the excretory pathway within the liver is believed to cause obstructive jaundice associated with primary biliary cirrhosis.

EXTRAHEPATIC CHOLESTASIS

This is caused by anatomical obstruction to flow of bile from liver to the intestine. The obstacle may be situated anywhere from the junction of right and left hepatic ducts to the termination of common bile duct in the duodenum. Atresia, stricture, choledocholithiasis, tumours of bile duct and pancreas, choledochal cysts and parasites have been implicated. Obstruction of extrahepatic duct results in an increase in serum bilirubin particularly the direct reacting type, the appearance of bile in the urine and passage of clay coloured stools. When total bilirubin level is above 3 mg/dL, the increase in both the direct and indirect reacting fraction parallel one another. With complete and persistant obstruction the serum bilirubin may plateau. In the fluctuating obstruction levels will change.

EFFECTS OF BILIARY TRACT OBSTRUCTION

PHYSICAL EFFECTS

The normal secretary pressure of bile is 120-250 mm of water. Following total bile duct obstruction, bile secretion will continue until CBD pressure rises to 170-220 mm of water after which secretion decreases. Cholesterol and phopholipid secretion is more readily reduced by high pressure than bile salt secretion making bile less lithogenic.

Complete obstruction of main extrahepatic bile duct of major segmental duct will normally lead to proximal dilatation. The lack of intrahepatic dilatation may be due to seconday hepatic fibrosis of co-existing alcoholic cirrhosis.

PAIN

Painless progressive jaundice is the classical hallmark of malignant biliary tract obstruction. But it is not uncommon to elicit a history of abdominal pain in these patients, the cause of pain being distention of gall bladder and bile duct or associated stretching of liver capsule in rapidly progressive obstruction.

PATHOLOGICAL CHANGES

BILE DUCTS AND CANALICULI

In biliary obstruction the canaliculi become dilated and microvilli distorted and swollen. Bile pigment thrombi may be seen in canaliculi and adjacent hepatocytes. In prolonged cholestasis, the canaliculi increase in length and tortuosity. Resorption of bile constituents from ductules leads to marked inflammatory reaction in the portal tracts with polymorphonuclear leucocyte

infiltrate. The hepatocyte of periportal zone shows disruption and eventually leading on to piecemeal necrosis. Experimental evidence shows that if obstruction is relieved within 2 weeks morphological changes are reversible.

CHOLANGITIS

Although the neutrophil associated with cholangitis is a chemical reaction associated with biliary obstruction and does not imply bacterial inflammation, in presence of biliary stasis, secondary bacterial colonization may produce the additional element of infective cholangitis although classically referred to as ascending cholangitis the actual mechanism for entry of bacteria into the unoperated biliary tract may not always be clear. Studies by McPherson et al (1982) showed that organisms are found in bile in approximately 1/3rd of patients with malignant biliary enteric anastamosis this rate may be higher. In another study Jackaman et al (1980) found that highest rate of biliary colonization were found in patients with choledocholithiasis and benign bile duct strictures where as may as 80% of patients had positive cultures.

ATROPHY

The characteristic effect of unilateral hepatic duct obstruction is atrophy of obstructed liver parenchyma with compensatory hyperplasia of unaffected segments of liver. A grossly hypertrophied left lobe palpable in association with unilateral obstruction may not produce sufficient hyperbilirubinemia in presence of normal contralateral lobe.

The practical importance of lobar atrophy in a surgical context lies in the fact that an atrophic liver lobe may be inadequate to support life following the resection of normal or hyperplastic

liver tissue and biliary drainage of such an obstructed lobe may also fail to produce resolution of jaundice.

BIOCHEMICAL EFFECTS

BILIRUBIN

Conjugated hyperbilirubinemia is the classical biochemical feature of obstructive jaundice. But prolonged partial obstruction with functional effects on hepatocytes may produce a mixed biochemical picture with elevated circulating unconjugated bilirubin.

ALKALINE PHOSPHATASE

Elevation of this enzyme is the most widely used and probably the most sensitive indicator. It may be the only biochemical indicator of incomplete of segmental obstruction. Acute obstruction of bile duct causes regurgitation of enzyme from biliary compartment and increase in hepatic synthesis.

PROTEIN SYNTHESIS

Liver occupies a central role in protein synthesis and quantitatively albumin is the most important protein synthesized by liver. However due to its long half life (20 days) only minimal changes occur to hepatocyte damage. Nonetheless the frequent association of biliary obstruction with malignancy and malnutrition causes hypoalbuminemia.

An active marker of hepatic protein synthesis, serum prealbumin is more valuable since it has half life of only 1.9 days. The most important aspect of protein synthesis relates to synthesis of coagulation factors II, VII, IX & X and its failure is due to failure to absorb vit K due to absence of bile salts from intestine.

LIPIDS

Cholesterol level may be elevated in biliary tract obstruction. A number of alterations in low density lipoproteins have been observed which are of no major functional importance.

CARBOHYDRATE METABOLISM

Abnormal glucose tolerance may be seen in patients with impaired liver function. But the malignant disease causing obstruction might be primary cause.

BILE SALT CIRCULATION

The enterohepatic circulation of bile salts is completely interrupted by total biliary obstruction. This may lead to gross elevation of serum bile acid levels. This has two important metabolic consequences. Firstly, due to the absence of bile salts in the intestine the small bowel microflora gets altered. Secondly, following external biliary drainage, the secretion of bile is under altered physiological drive.

ENDOTOXEMIA & RETICULOENDOTHELIAL FUNCTION

Endotoxin is a lipopolysaccharide derived from the cell walls of gram negative bacteria present in the gut. Normally only minute quantities of endotoxin enters the portal circulation and these traces are cleared by hepatic reticuloendothelial system. In obstructive jaundice the absence of bile salts from intestine causes increased formation of endotoxin by altered microflora and decreased clearance of absorbed endotoxin due to depressed reticuloendothelial cell function resulting in endotoxemia in more than 50% of patients. The bile salt absorption is also quiet fast in biliary obstruction probably due to increased vascular permeability. The circulating endotoxin causes pathological effects like renal

vasoconstriction, redistribution of intrarenal blood flow and activation of complement, leukocytes and platelets.

CHANGES AFTER RELIEF OF OBSTRUCTION

BILE SECRETION

Postoperative study of biliary secretion is done by the insertion of external percutaneous transhepatic drain. There is frequently a prompt and major cholestasis and bile volumes may exceed 4 liters per day. Failure to replace large volumes of fluid and electrolyte losses may result in dehydration and electrolyte depletion with a metabolic acidosis. The replacement of bile salts if desired may also be undertaken in the form of commercially available preparation.

During the first few days of biliary drainage the bile produced is of low bilirubin and bile salt concentration. This may be partly due to a slow return of impaired liver to normal function and also to loss of enterohepatic circulation of bile salts.

RECOVERY OF FUNCTION

In majority of case plasma bilirubin begins to fall promptly after insertion of a drainage catheter or an internal biliary bypass procedure and this is accompanied by clinical improvement. However, return of hepatocyte function is not instantaneous.

Assessment of liver function by serial antipyrene clearance measurement after relief of obstruction has shown that it takes about 6 weeks for it to return to normal values.

STRUCTURAL CHANGES

The reversal of structural changes in liver and biliary tract following decompression of obstruction is variable. Bile ducts which have beeen subjected to edema, inflammatory infiltration, cholangitis, and fibrotic changes are likely to retain some rigidity for considerable time after decompression.

As regards reversal of intra hepatic fibrotic changes following drainage it is difficult to obtain clear evidence since this would rely upon serial liver biopsies in asymptomatic patients so long as fibrotic changes remain short of true secondary biliary cirrhosis, they are reversible by adequate drainage. Even the portal hypertension secondary to such fibrosis may be improved with such drainage.

CLINICAL FEATURES

SYMPTOMS

Typically a patient with obstructive jaundice presents with dark urine, pale stools and pruritus of varying severity. Information regarding initial onset and whether clinical course is intermittent and associated with pain, fever and rigors must be short. Attack precipitated by fat intake can be relevant. An episode of cholangitis is recognized if jaundice is associated with pain, rigor and pyrexia. Jaundice without significant pain or pain radiating to back may indicate pancreatic pathology. However this is not certain and patients with gallstones an present with back pain whereas patients with extensive carcinoma of head of pancreas may present with typical history of biliary colic.

A fluctuating depth of jaundice is suggestive of intermittent obstruction as in periampullary carcinoma or temporary alteration of stone in the ampulla of vater. It is very rare in pancreatic cancer and cholangiocarcinoma. Weight loss, anorexia and pallor suggest malignancy of short duration. When these symptoms occur with painless jaundice, neoplasm of head of pancreas is likely. Pruritis may be present in all forms of jaundice and may either be progressive or fluctuate in intensity.

PHYSICAL EXAMINATION

GENERAL INSPECTION

The common stigmata of liver disease should be looked for – they are all indications of liver dysfunction. Jaundice is due to staining of tissues with bilirubin and possibly other pigments such as biliverdin. It is initially noticed in sclera. As jaundice progresses the skin becomes progressively more pigmented, spider naevi, which are vascular skin lesions supplied by central arteriole is occluded with a pinhead. Spider naevi usually occur in the region of superior vena cava – chest above the level of nipple, face, neck and arms. Palmar erythema is obvious and pronounced reddish flushing of palms. It particularly affects the thenar and hypothenar eminence and bases of fingers. Spontaneous bruising, echymosis and bleeding around venipuncture sites are well recognized signs of liver disease occurring due to abnormality in coagulation mechanisms. Long standing pruritis causing scratch marks all over the body can also be noted.

EXAMINATION OF LIVER

Palpation of liver should be combined with percussion to determine the upper and lower borders. The upper border of liver normally extends upto 5^{th} intercostals space. Auscultation

over the liver may give some evidence of underlying disease. An arterial bruit is evidence of hepatocellular carcinoma and venous hum in portal hypertension.

SPLENIC ENLARGEMENT

Splenomegaly can be detected by palpation commencing in the right iliac fossa and progressing towards the left hypochondrium. Splenic notch can sometimes be recognized on the anterior border of grossly enlarged spleen.

ASCITIS

Clinical confirmation of ascitis is achieved by eliciting shifting dullness on percussion or fluid thrill on palpating the flanks. Ascitis could be due to hypoalbuminemia of liver dysfunction, portal hypertension or manifestation of advanced malignancy either of liver or pancreas.

GALL BLADDER SIGNS

The finding of a palpable gall bladder in the presence of features of obstructive jaundice suggests malignant obstruction of the biliary tree (Courvoisier's Law). However failure to palpate gall bladder does not exclude the presence of malignant biliary obstruction. On the other hand it is possible to have a palpable gall bladder in the presence of gallstones where one stone obstructs the common bile duct and another is impacted in the Hartmann's pouch or cystic duct resulting in an empyema or mucocele of the gall bladder. An intermittently palpable gall bladder is suggestive of periampullary carcinoma.

EVIDENCE OF PORTAL HYPERTENSION

Portal hypertension is usually associated with hepatosplenomegaly and ascitis. Large dilated abdominal wall veins occur due to collateral circulation between the portal system and systemic veins.

DIFFERENTIAL DIAGNOSIS IN CHOLESTASIS

I. EXTRAHEPATIC CAUSES

1. STONES
   a) gallstones slipping into CBD
   b) gallstone in cystic duct and getting impacted onto CBD (Mirrizi syndrome)
   c) pancreatic calculus obstructing at the ampulla of Vater

2. STRICTURES
   a) malignant carcinoma of CBD
   b) Benign – surgical trauma
   c) primary sclerosing cholangitis

3. TUMOURS OF THE BILIARY TREE
   a) periampullary carcinoma
   b) carcinoma of head of pancreas
   c) cholangiocarcinoma

4. EXTRINSIC PRESSURE ON EXTRAHEPATIC BILIARY TRACT

a) Metastatic lymphnodes near the biliary tract by pressure and later by infiltration produce obstruction to biliary passages

b) primary lymphnodular disease involving the lymphnodes near biliary pathways – histiocytic non-Hodgkin's lymphoma

c) Metastatic involvement of the connective tissue of hepatic hilum causing extrinsic compression on bile ducts

5. MISCELLANEOUS CAUSES

a) parasitic occlusion of CBD – Schistosomiasis

b) Mycotic condition

c) Choledochal cysts

d) Hepatic artery aneurysm

II. INTRAHEPATIC CAUSES

1. INTRAHEPATIC STONE
2. INTRAHEPATIC BILIARY STRICTURES
3. KLATSKIN'S TUMOUR
4. BILIARY DYSPLASIA
    a) Congenital hepatic fibrosis
    b) Cystic disease of the liver
    c) Caroli's disease
5. CONGENITAL AND INFANTILE ATRESIA OF BILE DUCTS
6. ANEURYSM OF BRANCHES OF HEPATIC ARTERY
7. CYSTS OF THE LIVER

a) congenital

b) parasitic

8. PRIMARY AND SECONDARY MALIGNANCIES OF THE LIVER

CLINICAL CLASSIFICATION OF OBSTRUCTIVE BILIARY TRACT DISEASE

Classification proposed by Benjamin (1983) has proved useful in clinical practice. It recognizes four types of biliary obstruction. They are:

TYPE I: COMPLETE

Obstructive – producing progressive jaundice

Eg.:

a) Tumours of head of pancreas

b) Cholangiocarcinoma

c) Ligation of CBD

d) Parenchymal damage to liver

TYPE II: INTERMITTENT

Obstruction which produces symptoms and biochemical changes with or without jaundice

Eg.:

a) choledocholithiasis

b) periampullary carcinoma

c) duodenal diverticula

d) papillomas of bile duct

e) choledochal cysts

f) polycystic liver disease

g) intrabiliary parasite

TYPE III: CHRONIC INCOMPLETE

Obstruction with or without symptoms and biochemical changes eventually producing pathological changes in bile ducts of liver

Eg.:

a) strictues of CBD
    1) congenital
    2) traumatic
    3) post irradiation
b) stenosed biliary enteric anastamosis
c) stenosis of sphincter of Oddi
d) chronic pancreatitis
e) cystic fibrosis

TYPE IV: SEGMENTAL

Obstruction in which one or more anatomical segments of biliary tree may be obstructed. This in turn may be complete, intermittent or chronic incomplete.

Eg.:

a) traumatic (including iatrogenic)
b) hepatico choledocholithiasis
c) sclerosing cholangitis
d) cholangio carcinoma

INVESTIGATIONS

BIOCHEMISTRY

Biochemical features of cholestasis are:

1. Conjugated hyperbilirubinemia
2. Elevation of alkaline phosphatase, 5’ nucleotidase, gamma glutamyl transpeptidase. Th eenzyme 5’ nucleotidase is the most reliable since its level is not influenced by bone disease or alcoholism.
3. Minimal or no elevation of serum transaminases
4. Presence of bilirubin in the urine as conjugated bilirubin, which is water soluble and hence filtered by glomeruli
5. Elevation in serum cholesterol and bile acid levels although these are not routinely measured inpatients with cholestasis jaundice.

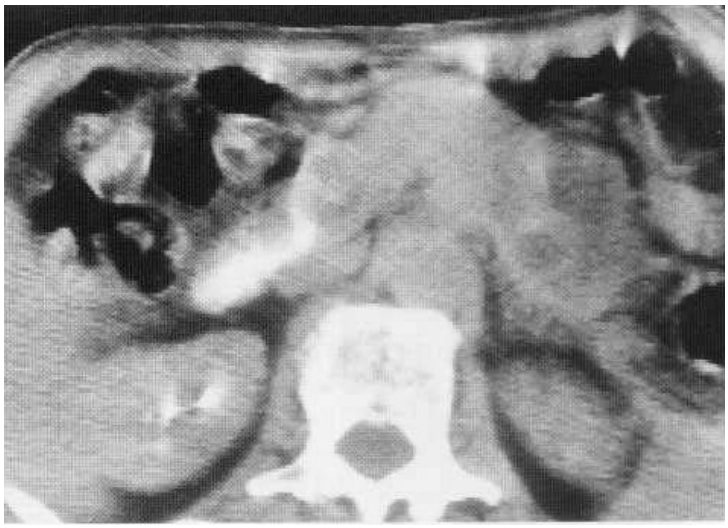
IMAGING TECHNIQUES

PLAIN ABDOMINAL AND CHEST SKIAGRAM

Calcification in the region of gall bladder indicates gall stone. Multiple areas of calcification in the region of the pancreas are helpful in diagnosing chronic calcific pancreatitis.

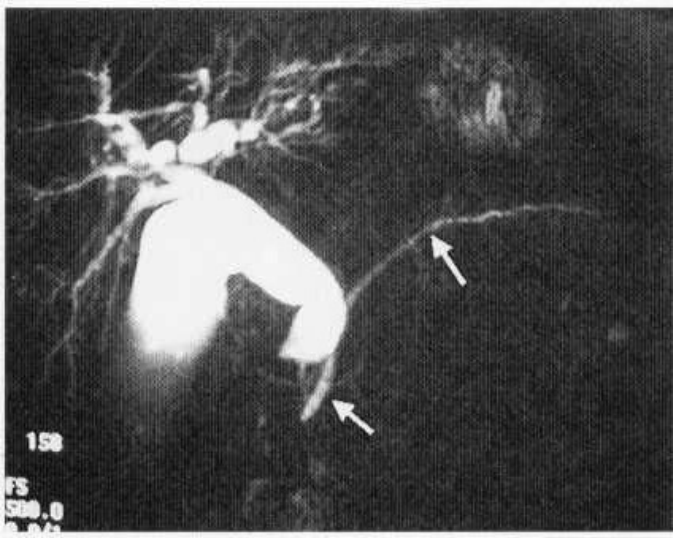
ULTRASONOGRAM

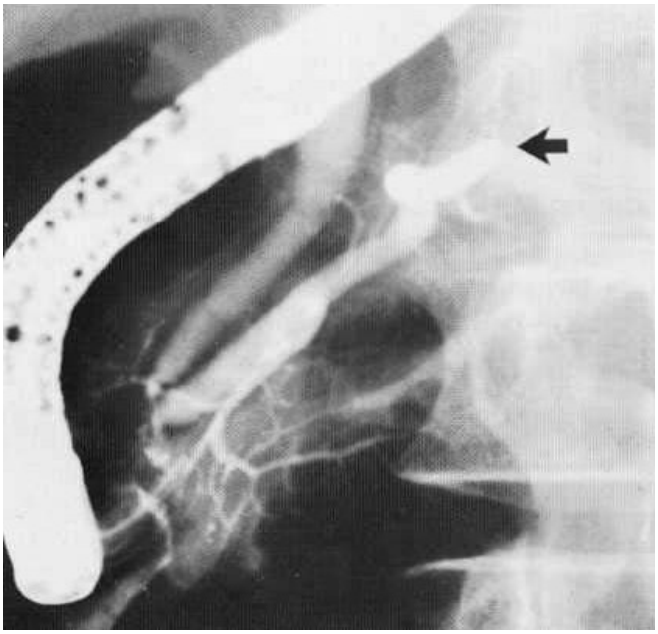
This is non-invasive and quick to perform, but requires experience in technique and interpretation. Extrahepatic biliary obstruction can be diagnosed by demonstration of dilated biliary radicals. In experienced hands the accuracy in diagnosing ductal dilatation is over 95%. In most iof the cases the cause of biliary obstruction can be traced by ultrasonogram. Enlargement of head of pancreas is suggestive of carcinoma. Difficulties in achieving a definite diagnosis arises principally with small lesions at the lower end of common bile duct and which is often obscured by gas in the duodenum or colon. As it does not involve radiation, it can be used safely in pregnancy.



CT - Ca. Tail of the Pancreas

MRCP – Ca. Ampulla of Vater





ERCP – Ca. Pancreas

ENDOSCOPIC RETROGRADE CHOLANGIO-PANCREATICOGRAPHY (ERCP)

Upper GI endoscopy with a forward or oblique viewing pan endoscope should be performed in jaundiced patients as significant gastrointestinal pathology is encountered in 25% of jaundiced patients. This is indicated when obstructing agent is lower down in the CBD. This is also ideal when ducts are not dilated or visualization of pancreatic duct or ampulla is required. It permits concomitant endoscopic examination and biopsy of lesions encountered during endoscopic examination. Certain lesions can be treated or palliated during this procedure like endoscopic stone removal, endoscopic nasobiliary drainage and stent insertion for inoperable malignant large bile duct obstruction. ERCP has very low morbidity due to pancreatitis (1%) and very low mortality (0.1%).

PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAM (PTC)

This is done by injecting the contrast material into the dilated biliary radicles through a cannula. This more useful when the obstruction is higher up in the bile or hepatic ducts.

CT findings in pancreatic carcinoma are:

a) Focal mass
b) Pancreatic atrophy/pancreatitis
c) CBD/PD dilatation
d) Vascular enhancement or displacement
e) Regional lymphadenopathy

MAGNETIC RESONANCE CHOLANGIO-PANCREATICOGRAPHY (MRCP)

It is a newer development in MRI. It provides multiplanar, cross-sectional, reconstructive image of pancreaticobiliary tree. It is purely diagnostic with no therapeutic intervention possible. It offers advantage over dynamic CT in suspected hilar cholangiocarcinoma and primary gall bladder carcinoma. Studies show promises with choledocholithiasis and malignant bile duct obstruction.

ENDOSCOPIC ULTRASONOGRAPHY

It is in the early stage of development. A useful modality for tumour exclusion when transabdominal utrasonography or CT has failed and high index of suspicion of carcinoma exists due to elevated tumour markers, prior to elevated tumour markers or ERCP. It can confirm tumour of 1.2 cm in head of pancreas.

LAPAROSCOPY

Should be routinely used by surgeons in all patients with jaundice. It gives direct visualization of underlying pathology and is valuable in staging hepatobiliary and pancreatic tumours. It avoids unnecessary laparotomy for patients with inoperable diseases.

ANGIOGRAPHY

Preoperative angiography is indicated in

1. history of previous major upper abdominal surgeries
2. doubtful resection on clinical and CT appearances
3. when it is anticipated to remove major vascular structures

Angiographic findings in pancreatic carcinoma are:

1. parenchymal hypovascularity
2. angulation of vessels
3. encasement of vessels (arterial/venous)
4. displacement of vessels
5. arterial neovascularity

FINE NEEDLE ASPIRATION CYTOLOGY

A fine needle of 21-23G is passed under guidance. The specimen is expressed on to microscopic side, smeared, fixed in 95% alcohol or autospray and stained by modified papanicolaou or other methods. Diagnosis can be made within 20 minutes of obtaining FNAC. Irregularly arranged clusters of cells exhibiting cellular pleomorphism, large vesicular nuclei and prominent nucleoli are seen. However such cytological appearances may sometimes fail to differentiate between adenocarcinoma of gland and lymphoma. Positive diagnosis can be obtained in 87-100% with few false positive results.

The earlier the tumour, the smaller it is. It then becomes difficult to obtain diagnosis by needle biopsy techniques. Complete resection remains the best biopsy. Every surgeon should be prepared to accept occasional benign biopsy report. This technique should be avoided in potentially respectable tumours with the theoretical possibility of seeding along needle tracks. It is to be noted that it could be on the other hand a very valuable method in elderly frail patients in whom a surgical palliation is being contemplated. It should also be avoided in a young relatively fit patient in whom a histological as opposed to cytological proof is mandatory prior to chemo or radiotherapy. The sensitivity ranges from 33-84%.

CARCINOMA OF PANCREAS

It includes carcinoma of the head proper and periampullary region. Almost all carcinoma of pancreas arise from the ductal epithelium. Only 1% arises from acini. The average age of patient is about 60 years, but carcinoma of ampulla the average age is about 5 years less. Males are more affected.

PATHOGENESIS

Incidence of carcinoma of the pancreas has risen steadily over the past 10 years. There are some factors, which can be considered as initiating or provoking carcinoma of pancreas. They are:

1. Cigarette smoking
2. Consumption of coffee
3. Diet rich in fat
4. Chemicals such as beta napthylamine and benzidine
5. Diabetes
6. Carcinogens in duodenal contents refluxing into the pancreatic duct
7. Alcohol consumption

PATHOLOGY

Adenocarcinoma is the predominant lesion often accompanied by extreme fibrous connective tissue stromal proliferation. The tumours may be mucinous of non-mucin secreting. Only 10% assume an adenosquamous pattern of extreme anaplasia with giant cell formation, numerous mitosis and bizarre pleomorphism. Only 5% arise in cyst and are termed cystadenocarcinoma.

Carcinomas of the ampulla of Vater are columnar cell adenocarcinoma. This neoplasm arises in duodenal papilla, in the ampulla of Vater or in the duodenal mucosa adjacent to the papilla there may be an area of pancreatitis in the head of pancreas. The primary lesion is so small that it is difficult to palpate. In such carcinoma, jaundice may not be progressive as recurrent sloughing of the central portion of the tumour will relieve obstruction of bile duct and jaundice becomes intermittent.

CLINICAL FEATURES

Carcinoma of the head of the pancreas usually presents with painless progressive obstructive jaundice. Progressive jaundice is usually associated with pruritis due to to the presence of bile salts in blood. The jaundice usually progresses steadily until the patient is almost green in colour. In case of periampullary carcinoma, the jaundice may be intermittent. Pain is not a marked feature. Patient may complain of dull and aching pain in the epigastrium. Pain is often relieved by sitting in hunched position and is aggravated by supine position. Eating may aggravate pain. Weight loss is the single most common symptom of carcinoma of the pancreas irrespective of the position of the tumour. Diarrhea with pale and foul smelling stool is sometimes a feature of periampullary carcinoma. There may be steatorrhoea due to enzyme deficiency.

On examination, jaundice is the main sign. A palpable distended gall bladder is detected in 60% of cases. Enlargement of liver is found in slightly more than half the cases. In carcinoma of head of pancreas it is often due to biliary obstruction.

Carcinoma of ampulla of Vater shows a few peculiar symptoms and signs. Pain is less frequent in this condition but when present is apt to be more colicky in nature. Jaundice is

intermittent. Chills and fever are not uncommon due to associated cholangitis. Hematemesis and melena occasionally occurs in late cases as a result of direct invasion of duodenal or gastric mucosa by tumour and portal hypertension secondary to splenic or portal vein compression by the tumour.

CHOLANGIOCARCINOMA

The reported autopsy incidence of malignant bile duct tumour ranges from 0.01-0.5%. There is slight preponderance of male (1.5:1). The age at presentation varies but the peak incidence is in sixth decade. The etiology of bile duct cancer is unknown. Cholangiocarcinoma is seen with increasing frequency in parasitic infestation of biliary tree, cystic disease of biliary tract, chronic typhoid carriers, and ulcerative colitis and sclerosing cholangitis.

PATHOLOGY

Tumours are best classified into the anatomical site of origin

1. Intrahepatic tumour from minor hepatic ducts
2. Proximal from right or left hepatic ducts, cystic duct and it confluence with CBD.
3. Middle from the distal common hepatic duct, cystic and its confluence with CBD.
4. Distal from the distal common bile duct and periampullary region.

Tumours of the minor hepatic ducts are often diffuse (multicentric) and difficult to differentiate from primary hepatocellular carcinoma. The gross appearance of cholangiocarcinoma assumes one of the three forms. They are:

1. Strictures
2. Nodular
3. Papillary

The majority of tumours are adenocarcinoma of varying origin. All cholangiocarcinomas have a special predilection for perineural spread and do not metastasise beyond the liver. The best prognosis is encountered after resection especially of distal and periampullary lesions.

CLINICAL FEATURES

The main presentation (90%) is with obstructive jaundice which is progressive and accompanied by itching and anorexia. Dull upper abdominal pain is a frequent symptom. Some patients present acutely with cholangitis. Physical examination reveals hepatomegaly. Anemia is present in patients with papillary tumours especially at the lower end of bile duct and periampullary region. It is caused by chronic blood loss. The feces of these patients have a characteristic silvery appearance due to combination of steatorrhoea and altered blood. A palpable gall bladder is present in patients with distal tumours.

PRE-OPERATIVE PREPARATION

1. All jaundiced patients must be kept in a good state of nutrition and hydration with supplemental intravenous fluids, elemental diet and multivitamins as deemed necessary. Renal failure due to hypovolemia is a tremendous hazard post-operatively and a continuous diuresis is maintained at all times. If the patient is grossly malnourished, a period of parenteral hyperalimentation both before and after operation may be of additional benefit.

2. Blood clotting deficiencies must be corrected. Anaemia is corrected by blood transfusions. Daily injection of Vit K is administered, preferably 4-5 days prior to

operation. Six units of fresh frozen plasma, six units of platelets and atleast six units of blood should be made available in operating room.

3. Cardiopulmonary function should be assessed by pulmonary function tests, chest X-ray and ECG. Smoking is prohibited. Intensive pulmonary physiotherapy, active mobilization and leg exercises are strongly encouraged post operatively.

4. Antibiotic prophylaxis should be given since there is impaired wound healing due to depressed immune function.

5. Nutritional status to be assessed and supported as there is impaired wound healing due to decreased fibroblastic activity and general protein and calorie malnutrition.

6. If patient is critically ill with one or more of the following parameters,
    a) highly elevated serum bilirubin (>12 mg%)
    b) sepsis
    c) hepatorenal failure
    d) severe cardiopulmonary disease
    e) malnutrition

    a percutaneous transhepatic biliary drainage or endoscopic decompression should be attempted to tide over the patient for 2-3 weeks before major surgery. If the technique of percutaneous biliary drainage or endoscopic stenting is not available, a simple cholecystectomy or T-tube drainage of CBD may be undertaken.

TREATMENT OF MALIGNANT OBSTRUCTIVE JAUNDICE

Treatment can be either

1. Curative
2. Palliative

CURATIVE TREATMENT

Surgery is now considered as the gold standard for treatment of malignant obstructive jaundice against which all other new modalities are considered. Halsted performed first curative and successful resection of periampullary carcinoma at John Hopkins Hospital in 1898. He performed local resection of ampullary tumour. Presently standard resection for periampullary carcinoma and head of pancreas tumours involves a pancreaticoduodenectomy, first performed successfully by Kausch in 1909 and popularized by Whipple 1935. The gall bladder, CBD, entire duodenum, head of pancreas, pancreas upto the level of superior mesenteric vein, pylorus and distal stomach are resected. Restoration of gastrointestinal continuity utilizes the proximal jejunum, brought out through the transverse mesocolon for pancreaticojejunostomy, hepaticojejunostomy and gastrojejunostomy. The standard Whipple resection remains the classic thrrapy for these tumours and can be successfully performed in experienced hand swith mortality less than 5%. A modification of standard Whipple resection, the pylorus preserving pancreaticoduodenectomy has gained popularity in recent years. This modification eliminates gastric resection and leaves a 2cm cuff of duodenum for enteric reconstruction as duodenojejunostomy.

PALLIATIVE SURGERY

Palliative surgery for periampullary carcinoma is performed in patients with unresectable disease discovered at the time of laparatomy or in patients with prohibitive risk for resectional

therapy (advanced age, limited cardiopulmonary reserve and also poorly alleviated non-operatively).

1. Relief of jaundice, pruritis and impending cholangitis: Biliary tract decompression can be done either by cholecystojejunostomy or by hepaticojejunostomy (each with diverting enterostomy) depending on whether the cystic duct is widely patent and is in full communication with the biliary tree proximal to the obstructing cancer.

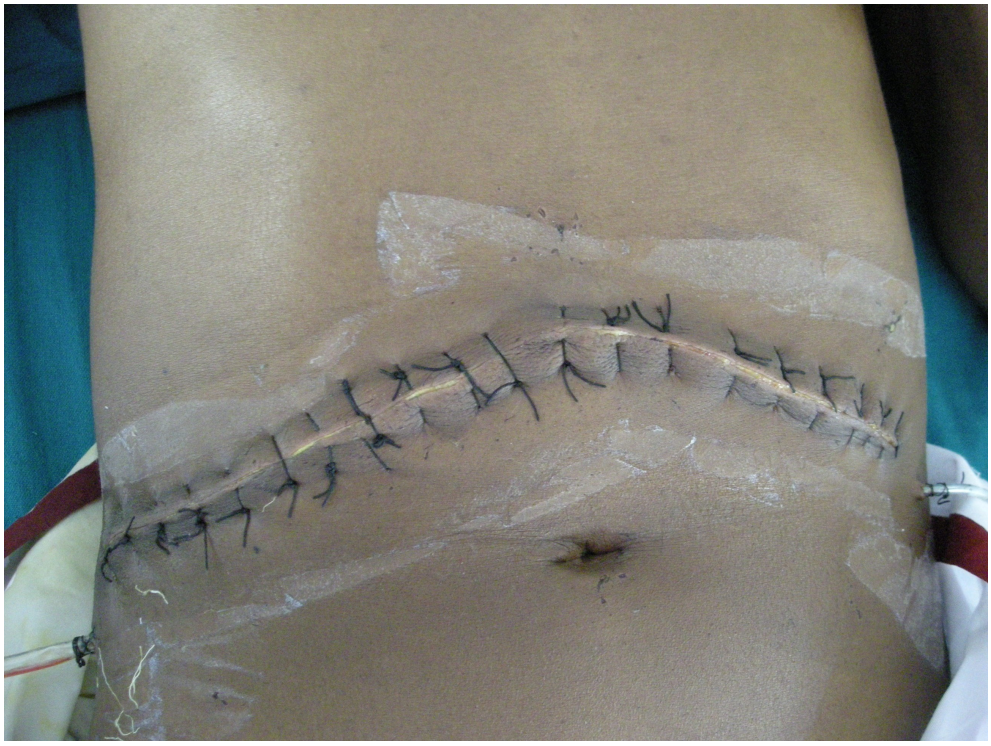
2. Relief of duodenal obstruction: If the patient lives for more than few months, duodenal obstruction usually occurs. It is therefore advisable to perform a gastrojejunostomy at the primary operation.

NON OPERATIVE MANAGEMENT

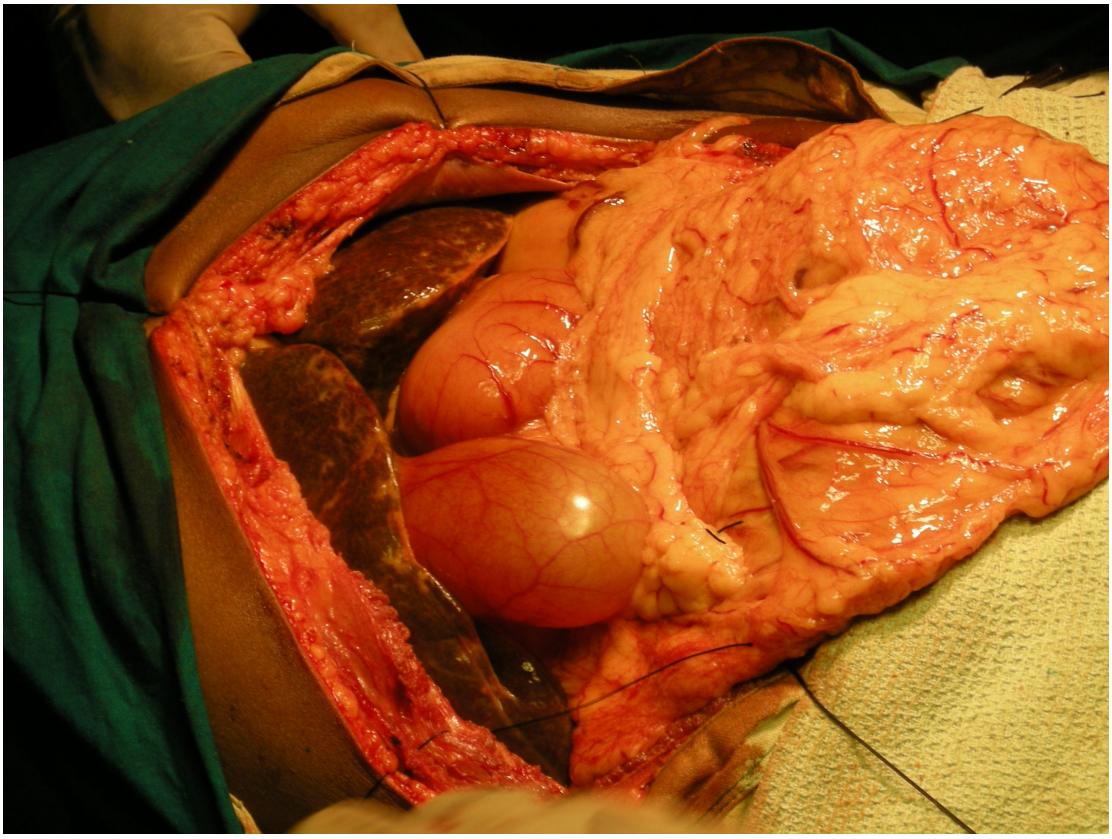
When a patient is unfit or refuses surgery an alternative method of palliation of the jaundice is by endoscopic sphincterotomy and placement of biliary stent. This approach does not relieve any additional obstruction, which may be present. If patient survives for more than a few months, recurrent cholangitis associated with stent blockage is a problem that necessitates regular endoscopic removal and replacement of the stent.

Percutaneous transhepatic placement of internal expandable metal stent is being tried by inteventional radiologist and offers yet another option for palliation of the jaundiced patient with malignant biliary tract obstruction.

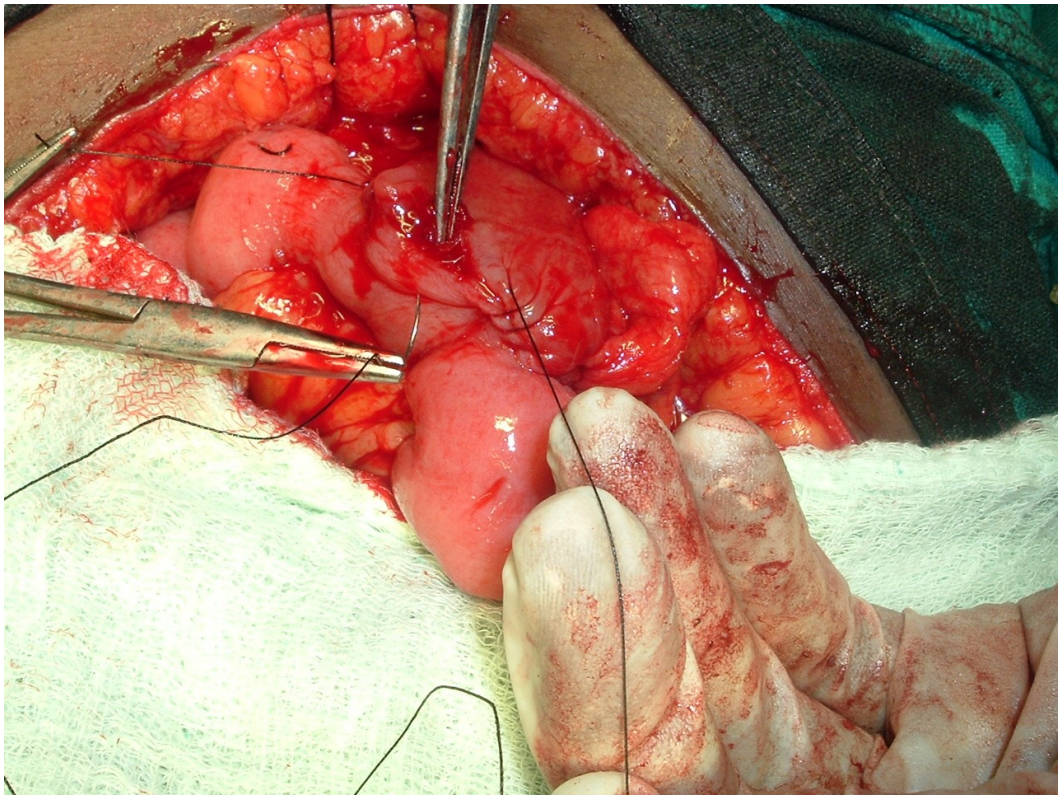
Rooftop Incision



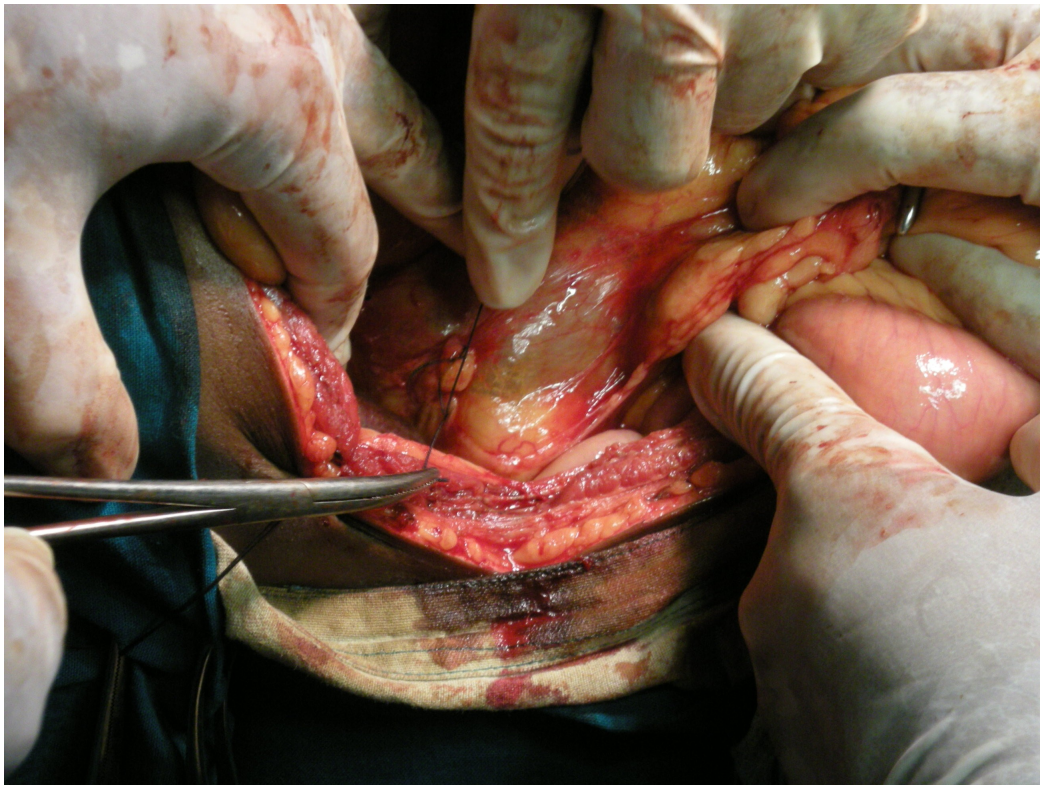
Exposure of the Abdominal Cavity



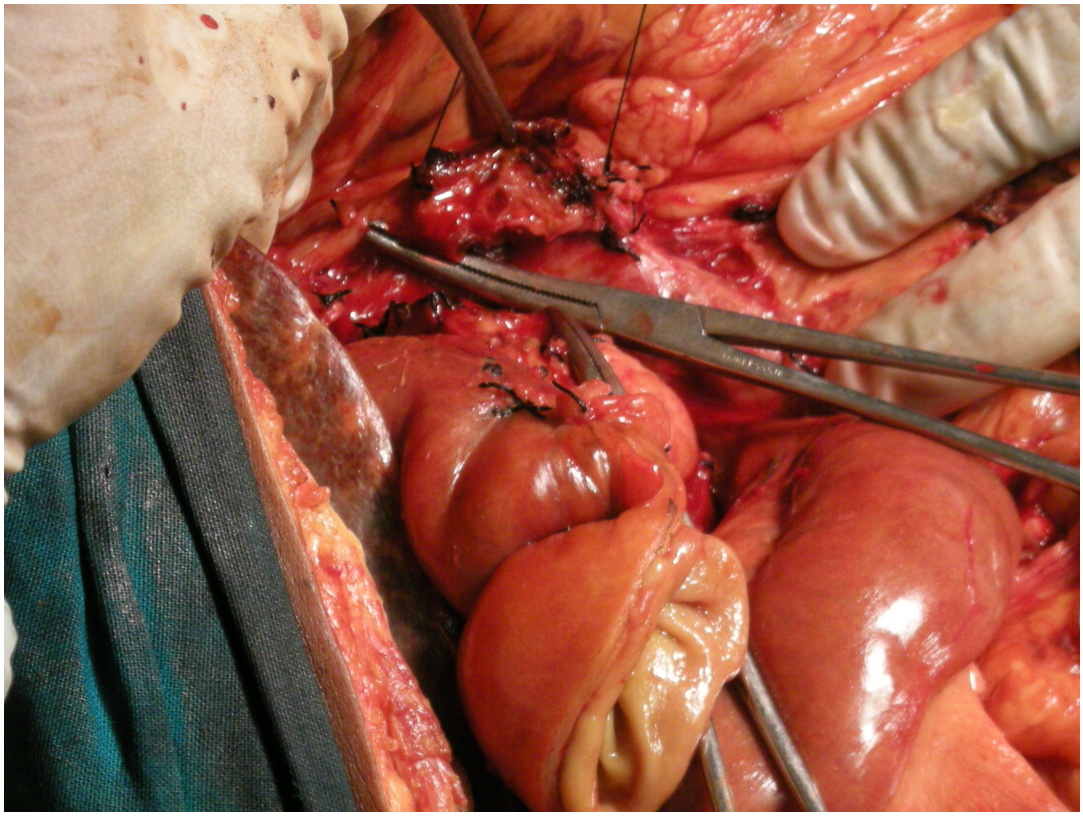
Gastro-Jejunostomy in progress



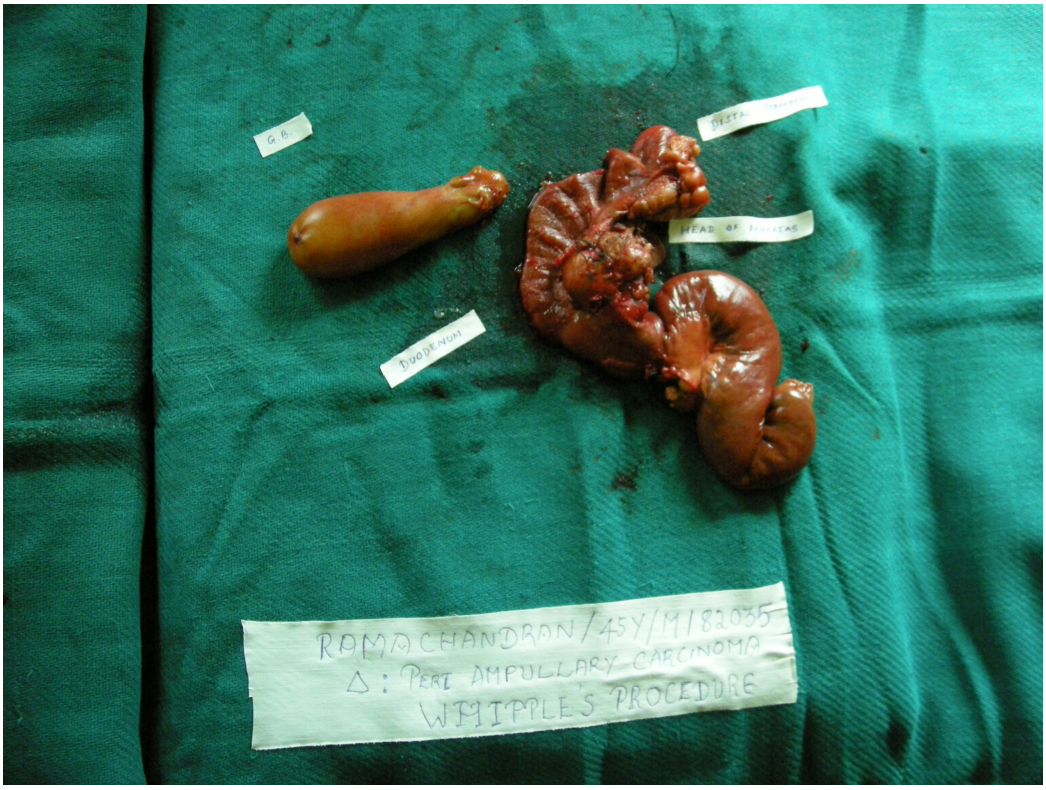
Choledocho-Jejunostomy in progress



Pancreatico-Jejunostomy in progress



Resected Specimen



TREATMENT OF CHOLANGIOCARCINOMA

Resection is the best method of treatment and is indicated for all operative tumours in fit individuals. The reported respectable rate varies but averages 20%. The benefits of resection are:

1. The possibility of cure or long term survival especially for distal bile duct tumours
2. Resection provides the best form of palliation in terms of duration and freedom from infective complications.

The surgical procedure depends on the location of tumour. For hilar lesions, an anterior segmentectomy IV provides good access to confluence, allowing good clearance proximal to the tumour and facilitates hepaticojejunostomy. When the tumour extends along the right or left duct with extension to respective lobe, the resection includes a lobectomy in continuation with main tumour mass. Middle tumours are excised from just below the confluence down to the duodenum together with associated pericholedochal lymphnodes. The surgical treatment of periampullary tumours is pancreaticoduodenectomy. The results of hepatic transplantation for cholangiocarcinoma (diffuse intrahepatic type) have been disappointing.

PALLIATIVE SURGERY

If tumour is inoperable, a bilio-enteric bypass is performed. Anastamosis of Roux loop to segment III duct using the round ligament approach gives the best results for inoperable hilar lesions. Longmire operation in which anastamosis of the segment III duct to Roux loop of jejunum after left lateral segmentectomy and Smith operation used to be done earlier, but there is no added advantage to these procedures. A cholecystojejunostomy is performed for inoperable distal tumours. A gastrojejunostomy is added if duodenal obstruction is present or considered imminent in patient with periampullary tumour.

NON OPERATIVE MANAGEMENT

In patients who are considered inoperable as preoperative assessment and those who are too old and frail, palliation of jaundice is achieved by percutaneous transhepatic or endoscopic stenting. The endoprosthesis has to be large 8-10 FG and may require replacement if it becomes blocked. Recently self-expandable stainless steel wire endoprosthesis have been introduced in management of patients with malignant biliary strictures. Other causes of malignant obstructive jaundice are due to extrinsic compression on the biliary tract by tumours, Metastatic lymphnodes near the biliary tract by pressure and later by infiltration and primary lymphonodular disease involving lymph nodes near biliary pathways. Treatment is primarily to relieve obstructive jaundice and troublesome pruritis and steatorrhoea.

CHAPTER IV

AIM OF STUDY

1. To analyze the incidence of causes of malignant obstructive jaundice in our hospital.
2. To analyze the age and sex distribution.
3. To study various clinical presentations.
4. To evaluate various management modalities.
5. To analyze complications associated with obstructive jaundice.
6. To evaluate the histopathology of resected specimen.

CHAPTER V

MATERIALS AND METHODS

DESIGN

This is a prospective descriptive study. Study population has been selected after necessary exclusion criteria have been applied.

SETTING

The study is done at a tertiary care centre namely, Govt. Rajaji Hospital, Madurai in various General Surgery units and the Dept. of Surgical Gastroenterology. The period of study is from June 2006 to November 2008.

POPULATION / PARTICIPANTS / SAMPLE SIZE

A random selection of 50 patients admitted in surgical wards within June 2006 to Nov 2008 has been done.

INCLUSION CRITERIA

1. Patients with malignancies of hepatobiliary system or pancreas producing obstructive jaundice
2. Patients with malignancy outside the hepatobiliary system or pancreas producing infiltration of biliary tree or secondaries in porta hepatis.

EXCLUSION CRITERIA

1. Patients with benign causes of obstructive jaundice.
2. Patients with hemolytic and hepatocellular jaundice.

CHAPTER VI

PROFORMA

Name	:	
Age	:	
Sex	:	
Address	:	
IP Number	:	
Unit	:	
Date of Admission	:	
Date of Surgery	:	
Date of Discharge/Death	:	
Duration of illness	:	
Symptoms	:	Weight loss, diarrhea, jaundice, bleeding per rectum, melena, abdominal pain, anorexia, steatorrhoea, dark urine, constipation, belching, vomiting, hematemesis, abdominal distention, pruritis
Signs	:	Icterus, hepatomegaly, palpable gall bladder, tenderness, ascitis, palpable spleen, abdominal mass, supraclavicular nodes and lymphnodes
Past History	:	Chronic calcific pancreatitis, diabetes mellitus, previous surgery
Personal History	:	Exposure to chemical carcinogen, alcohol, tobacco, coffee, dietary habits
Family History	:	Pancreatic, bowel or other malignancies
Lab Investigations	:	USG, CT scan, ERCP/PTC, Angiography, liver function tests, RBS, BUN, serum creatinine
Assessment of Surgical Risk	:	
Preoperative Decompression Procedures	:	
Surgical Procedure	:	Exploratory laparotomy, drainage, palliative resection and curative resection

Blood Transfusion given	:	
Per-Op Findings	:	
Metastasis	:	Regional lymphnodes, liver, peritoneum and other sites
Post-Op Complications	:	
Histopathological Report	:	
Mean Hospital Stay	:	

CHAPTER VII

RESULTS OF ANALYSIS

A total of 50 cases of malignant obstructive jaundice were included in the study. These cases were admitted in the Govt. Rajaji Hospital, Madurai between June 2006 and November 2008. Going through the statistics it is found that there are a substantial number of patients with malignant obstructive jaundice.

Out of the 50 patients studied in detail there were 31 males and 19 females. Age analysis of these cases showed that maximum incidence is between 41-50 age group.

Out of the 50 patients, 15 were diabetic patients whose diabetic status was kept under control with varying measures from adjustment of diet to insulin administration. 15 patients gave a past history of jaundice.

In our study, jaundice and dark coloured urine was complained by almost all patients followed by anorexia and weight loss. A palpable gall bladder was complained in 29 patients. Presentation with mass and ascitis are common when compared to western studies.

Among the malignancies causing obstructive jaundice carcinoma of head of pancreas is the commonest cause, followed by periampullary carcinoma, cholangiocarcinoma and carcinoma of the gall bladder. Other miscellaneous causes include secondaries at porta hepatis from stomach and gastrointestinal tract and also malignancies producing extrinsic compression of bile duct.

Of various risk factors in pancreatic carcinogenesis, we have made an in depth analysis of tumours of pancreas with chronic calcific pancreatitis. The relationship with familial chronic pancreatitis (FCP) is now well documented (Comfort and Steinberg, 1952) and was found to be seen in 1/3rd of patients developing pancreatic cancer. Castleman et al in 1972 stated that first degree relatives of patients with familial calcific pancreatitis run an increased risk of cancer even without chronic pancreatitis. In our study, the risk rate was 24%.

Operative procedures done for patients with malignant obstructive jaundice are many. Cholecystojejunostomy and gastrojejunostomy leads the list with 29 patients undergoing the procedure. Classical Whipple's resection was done in 10 patients and modified Whipple's resection in 4 patients. One patient was subjected to Choledochojejunostomy since cystic duct insertion was very low and there was dilated CBD. Segment III bypass and hepaticojejunostomy were done in three patients with Klatskin's tumour. In 4 patients no palliative surgeries were possible and only stenting was done. No surgical intervention was done in 3 patients as they refused surgery and due to poor general condition.

When laparatomy was performed, it was found that regional lymphnodes were affected in majority (76.5%) followed by liver metastasis and peritoneal deposits. Very few had metastasis to pelvic regions, lungs and brain.

Complications following Whipple's procedure were low. There were 4 cases of fistula, 2 cases of pancreatitis and 2 cases of wound infection. Two patients died in the post-operative period (mortality within 30 days of surgery). One patient died due to myocardial infarction and one patient due to life threatening infection and cholangitis.

In four cases of obstructive jaundice due to extension of growth from stomach into the region of terminal CBD, cholecystojejunostomy with gastrojejunostomy was possible for palliation of jaundice and duodenal obstruction. The remaining 2 cases had infiltration from the growth upto porta hepatic where no decompression was attempted.

None of the common hepatic duct or common bile duct growths were resectable due to local infiltration to the vital vasculature around porta hepatis. Out of 9 cases with such growths, four were treated with placement of stent through the growth into the dilated intrahepatic biliary tree.

In 5 cases of carcinoma of gall bladder, the growths were very advanced with infiltration of hepatic ducts and metastasis in liver. Two patients were treated with placement of stent through the growth into the dilated biliary tree. And in other three patients no surgery was possible.

Wound infection was reported in 9 patients followed by cholangitis in 6 patients, which was treated by antibiotics and removal of collection and proper stressing. There were 6 cases of delayed gastric emptying which were treated with nasogastric suction.

There were 8 deaths in this group. Most of the patients died due to the threatening cholangitis and renal failure.

Histopathology reports of patients subjected to Whipple's resection showed most of them to be moderately and poorly differentiated adenocarcinoma. There was well differentiated histology in only 5 cases.

CHAPTER VIIII

DISCUSSION OF ANALYSIS

Surgery for obstructive jaundice is fascinating because of wide variety of etiological factors, varying number of surgical methods and technical challenges in reaching pancreas and biliary tree.

The observation that many of the patients were diabetic show they stand a higher risk of development of pancreatic malignancy. The study of relationship of chronic calcific pancreatitis patients with carcinoma of pancreas is well documented in 1/3rd of patients developing pancreatic cancer. Castleman et al in 1972 stated that first degree relatives of patients with familial calcific pancreatitis run an increased risk of cancer even without chronic pancreatitis. In our study the risk rate was 22.5%.

Whether to go for a radical surgical procedure like Whipple's resection or choose a simple bypass procedure in cases of malignant lesions of pancreas producing obstructive jaundice is highly debatable. The hospital mortality for pancreaticoduodenectomy has decreased substantially and long term survival has improved significantly. In mid 1990 pancreaticoduodenectomy can be performed at regional referral centres with an operative mortality of 1-2%. Five year survival between 20-30% for patients with resected pancreatic cancer. Recent data suggests that quality of survival is better with standard operations and best with pylorus preserving pancreaticoduodenectomy (PPPD). However debate will continue.

In 1993 Geer and Brennan reported 130 patients undergoing standard pancreaticoduodenectomy with actual survival rate of 21%. In John Hopkins hospital, 201 patients undergoing pancreaticoduodenectomy showed actual 5-year survival rate as 21%. This study also demonstrates that survival has improved from decade to decade. In sharp contrast the Mayo clinic group reports a hospital mortality of 3% and a 5-year survival rate of 6.8%.

The factors which appear to influence survival include negative resection margins, tumour diameter more than 5 cm, DNA content measured by image cytometry. In patients with Hopkins series in whom the DNA content was diploid, 5 year survival was 39% and when tumour was aneuploid, 5 year survival was only 8%. Lymphnode status is an important factor that has bearing on prognosis. In series from Hopkins 144 patients were node positive and 5-year survival in this group was 14%. Thus positive lymphnodes is clearly a negative prognostic finding. Prognostic predictive values of other factors have been arrived at in yet another study in univariate but not in multivariate analysis. These include histological differentiation, gender of patient, operative time and number of blood transfusions. Survival following pancreaticoduodenectomy has improved over the past several decades. There are multiple reasons for this. Between 1970 and 1980, a decrease in the number of blood transfusions clearly played a contributory role. Positive resection margins were common in 1970s than 1980s and 1990s and this played an important role. Of late from these records it is evident that Whipple's resection is a better form of surgical treatment atleast in cases of periampullary carcinoma in experienced hands.

Treatment of cholangiocarcinoma, especially hilar carcinomas are disappointing. The place of resection for attempted cure of patients with tumour at confluence of bile duct is strongly

debated. Thus Longmire was able to resect only 6 of 33 lesions and tumour excision combined with hepatic resection. Similarly Smith (1981) treated 33 cases in 33 years and excised only 5. in none there was hepatic resection. Longmire (1973) in anatomical and clinical study recognized that involvement of vessels was the limiting factor to resection in many instances. Risk of drainage operation in treatment of biliary tract obstruction is high with mortality of 20% or more. Indeed even nonoperative endoscopic or percutaneous intubation methods for the relief of obstructive jaundice are associated with significant morbidity and 30 day mortality of 20%. Majority of patients with cholangiocarcinoma will not be suitable for resection and may be ill or old or have concomitant disease. The option open in the management are either no treatment at all or some form of biliary decompression either by means of biliary enteric anastamosis or by one of trans-tumoural tube allowing relief of biliary obstruction. Alterative methods of tubal drainage (such as placement of transhepatic endoprosthesis or percutaneous transhepatic drainage). They have potential for serious infection and or PTBD 30 day mortality of atleast 20% is reported. Evander and colleagues in a series of 53 patients subjected 40 to some form of biliary drainage, majority by percutaneous transhepatic technique. The median survival was only 2.5 months. It is clear that results of nonoperative techniques for biliary decompression in biliary cancer have yet to show improvement over surgical approach. However with liberal use of round ligament approach 28 patients were operated on with mortality of 2.1%. Recent experiences in France (Bismuth 1988) reveal a good quality palliation with operable mortality of only 7%. The results are better than those achieved by nonsurgical methods.

In 7 cases of cholangiocarcinoma in our study, treatment was disappointing – only 3 (4.8%) were subjected to segment III bypass and 4 patients were palliated with transtumoural stenting. No operative or palliative procedures were attempted in 3 patients.

CHAPTER IX

SUMMARY AND CONCLUSION

1. There is a significant increase in the incidence of malignant obstructive jaundice
2. Majority of tumours are in the head of pancreas (54%)
3. The maximum of age incidence is between 41 and 50 years (50%)
4. Male : Female ratio is 3:2
5. Resectability rate was 20.8% with 10 patients undergoing Whipple's resection or its modifications
6. Chronic calcific pancreatitis is a premalignant condition
7. A 26% incidence of CCP with carcinoma of head of pancreas was noted.
8. 100% of patients complained jaundice, weight loss and anorexia.
9. A palpable gall bladder was noted in 58% of the cases compared to 30-35% reported in western literature.
10. A palliative Cholecystojejunostomy with gastrojejunostomy tops the list of operative procedures
11. Mortality due to palliative procedures was 7% and morbidity patterns of wound infection is 9.2%, cholangitis is 5% and delayed gastric emptying is 6%.
12. Median hospital stay for palliative procedures was 21 days.
13. Mortality rate following Whipple's procedure was 7.8%
14. Only palliative procedures are possible in Klatskin's tumour like segments III bypass (4.2%) and transtumoural stenting (6.2%)
15. Moderately differentiated adenocarcninoma was the histology in 40%, poorly differentiated adenocarcinoma 30% and well differentiated in 30%.

CHAPTER X

TABLES AND CHARTS

Table 1. Age distribution of cases in study population

Age Group	Number	Percentage
31-40	5	10%
41-50	25	50%
51-60	12	24%
61-70	8	16%

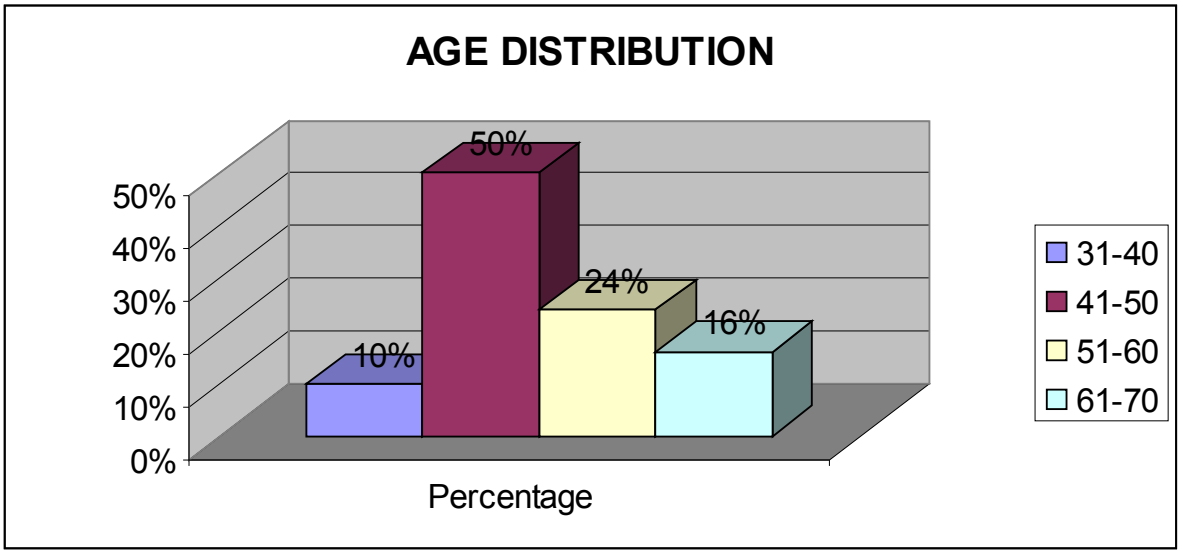


Table 2. Sex Distribution

Sex	Number	Percentage
Male	31	62%
Female	19	38%

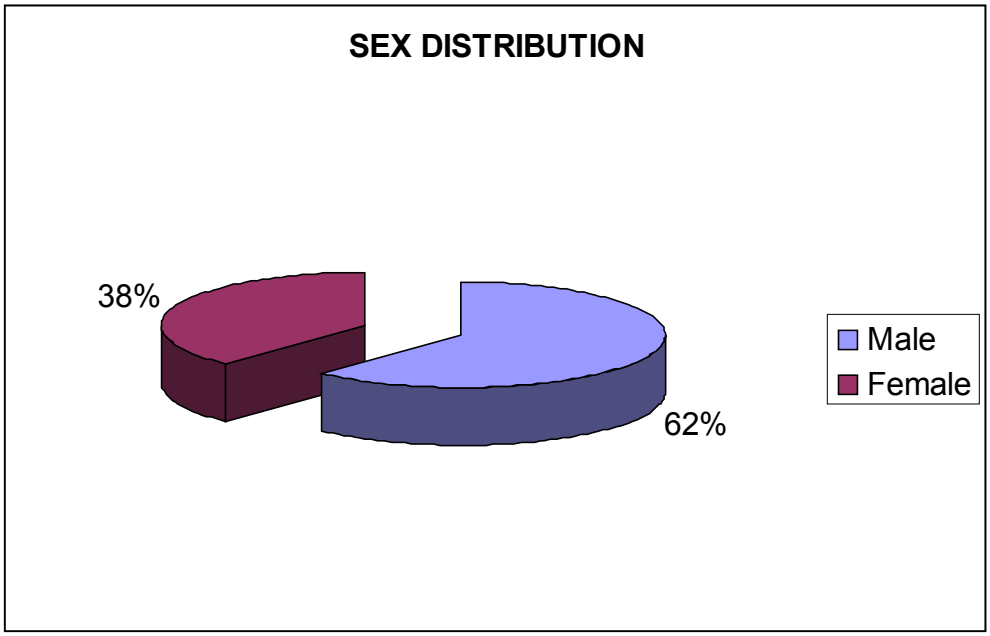


Table 3. Tumour Types

Tumour types	Number	Percentage
Carcinoma of head of pancreas	27	54%
Periampullary Carcinoma	12	24%
Cholangiocarcinoma	5	10%
Carcinoma gall bladder	2	4%
Miscellaneous	4	8%

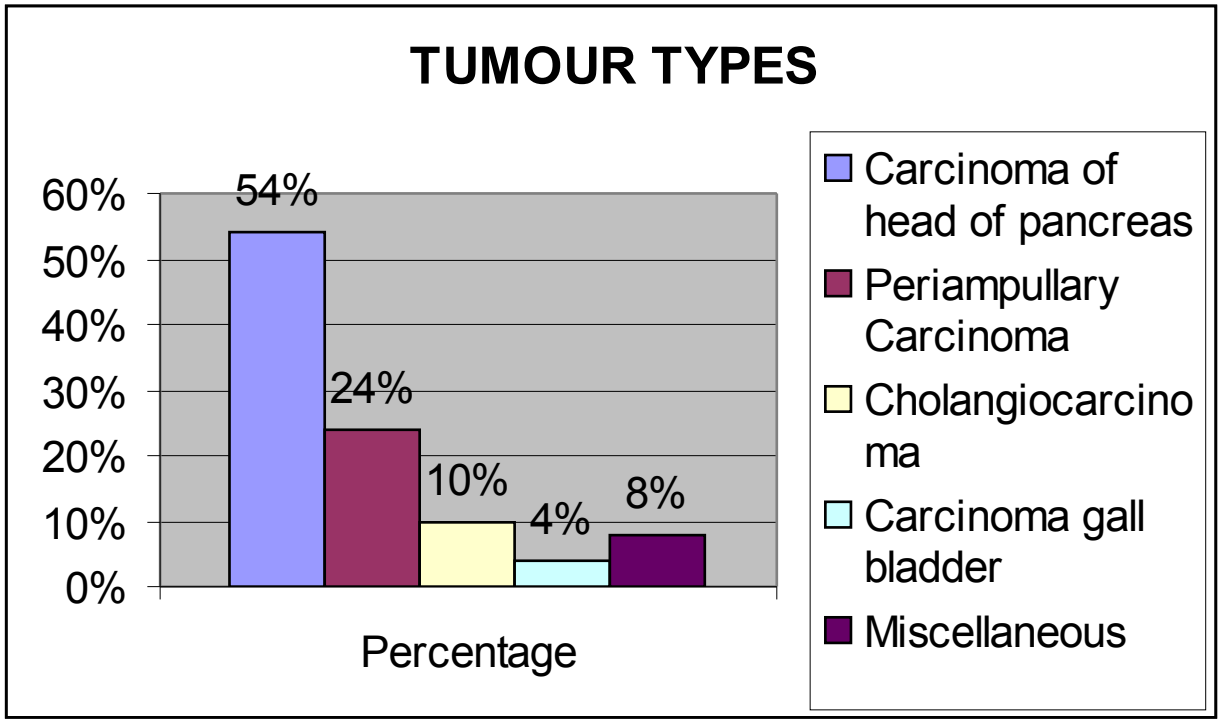


Table 4. Relationship of carcinoma head of pancreas with chronic calcific pancreatitis

Carcinoma of head of pancreas	Number	Percentage
With chronic calficic pancreatitis	7	25.9%
Without chronic calcific pancreatitis	20	74.1%

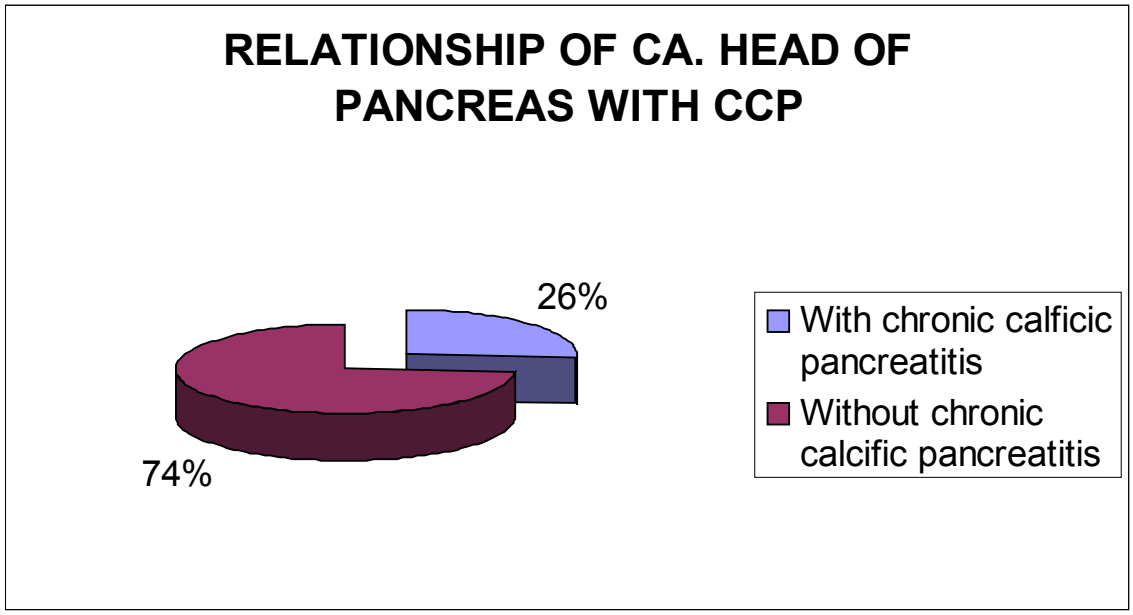
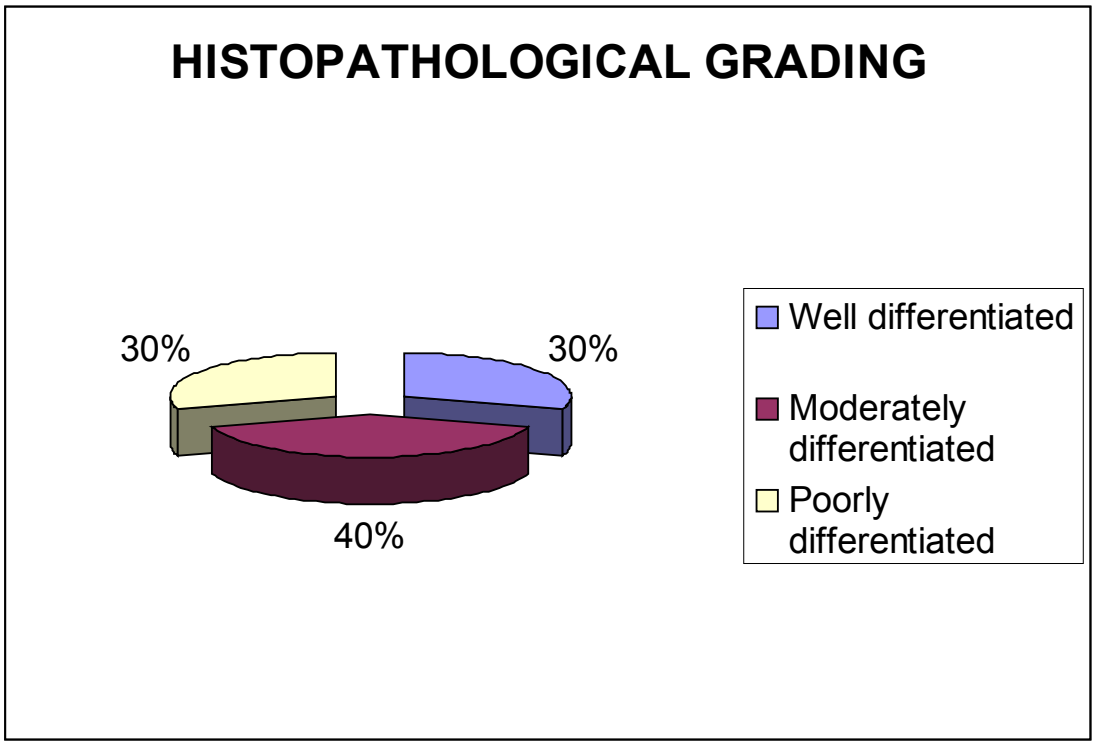


Table 5. Histopathological Grading of Tumours resected by Whipple's Procedure

Histopathological Grading	Number	Percentage
Well differentiated	3	30%
Moderately differentiated	4	40%
Poorly differentiated	3	30%



CHAPTER XI

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MASTER CHART

Sl. No	Name	IP No.	Age	Sex	Clinical Features	Palp. GB	Ca. Type	CCP	Grading	Surgery	Post-op Complication	Status at 30 days
1	Selvakumar	452538	65	M	J / DU / WL	+	PC		–	Palliative	Uneventful	Alive
2	Shanmugam	454460	54	M	J / DU	+	HP	+	–	Palliative	DGE	Alive
3	Kamatchi	462152	45	F	J / DU /WL	+	M		–	NIL	Uneventful	Dead
4	Valarmathy	467817	57	F	J / DU /WL	+	HP	–	–	Palliative	CI	Alive
5	Velmurugan	475316	42	M	J / DU	+	HP	+	–	Palliative	WI	Alive
6	Ulaganathan	480613	65	M	J / DU /WL	–	HP	+	–	Palliative	Uneventful	Alive
7	Chinnathai	494261	46	F	J / DU /WL	+	PC		Well Diff.	Whipple	Pancreatitis	Alive
8	Narayanan	74325	44	M	J / DU	–	HP	+	–	Palliative	CI	Dead
9	Muthulaxmi	76183	55	F	J / DU	–	CC		–	Palliative	WI	Alive
10	Sethupandi	72505	45	M	J / DU /WL	–	HP	–	–	Palliative	DGE	Alive
11	Kattaiyan	77430	32	M	J / DU /WL	–	CGB		–	Stenting	Uneventful	Dead
12	Vijaya	81952	47	F	J / DU	+	PC		–	Palliative	Uneventful	Alive
13	Amsavalli	90017	47	F	J / DU	–	HP	+	Poor Diff.	Whipple	WI	Dead
14	Udaiyappan	119871	62	M	J / DU /WL	–	HP	–	Poor Diff.	Whipple	Fistula	Alive
15	Jayalaxmi	128392	42	F	J / DU /WL	+	HP	+	–	Palliative	Uneventful	Alive
16	Paramasivam	140599	57	M	J / DU /WL	+	M		–	Stenting	DGE	Alive
17	Kandhasamy	183033	48	M	J / DU /WL	+	PC		–	Palliative	WI	Alive
18	Laxmi	172923	45	F	J / DU /WL	+	HP	+	Well Diff.	Whipple	Fistula	Alive
19	JeyaShree	15018	43	F	J / DU /WL	–	CC		–	Palliative	CI	Dead
20	Pavithra	16681	52	F	J / DU	–	HP	–	–	Palliative	WI	Alive
21	Sakthivel	17723	44	M	J / DU	+	PC		–	Palliative	Uneventful	Alive
22	Rajendran	20275	49	M	J / DU /WL	+	PC		–	NIL	Uneventful	Dead
23	Madasamy	21404	56	M	J / DU /WL	+	HP	+	Mod. Diff.	Whipple	WI	Alive
24	Ganesan	21948	34	M	J / DU	–	PC		–	Palliative	Uneventful	Alive
25	Annalaxmi	33049	50	F	J / DU	+	HP	–	–	Palliative	DGE	Alive
26	Alamelu	58395	43	F	J / DU /WL	–	CC		–	Palliative	Uneventful	Alive
27	Mohd. Ali	50339	58	M	J / DU /WL	–	HP	+	Mod. Diff.	Whipple	Pancreatitis	Alive
28	Rajangam	60438	46	M	J / DU /WL	+	HP	+	–	Palliative	Uneventful	Alive
29	Selvi	73938	70	F	J / DU /WL	+	PC		–	Palliative	CI	Alive
30	Chinnasamy	93848	59	M	J / DU	–	PC		–	Palliative	Uneventful	Alive

Sl. No.	Name	IP No.	Age	Sex	Clinical Features	Palp. GB	Ca. Type	CCP	Grading	Surgery	Post-op Complication	Status at 30 days
31	Raman	101119	45		J / DU /WL	–	HP	+	Mod. Diff.	Whipple	Fistula	Dead
32	Periyavalli	120397	47	F	J / DU /WL	–	PC		–	Stenting	Uneventful	Alive
33	Mohd. Mathar	145940	55	M	J / DU /WL	–	CC		–	Palliative	WI	Alive
34	Pattammal	194848	42	F	J / DU /WL	+	HP	–	–	NIL	Uneventful	Dead
35	Pushpam	19384	33	F	J / DU /WL	+	CGB		–	Stenting	DGE	Alive
36	Ponnusamy	28437	65	M	J / DU /WL	+	HP	–	Poor Diff.	Whipple	Uneventful	Alive
37	Ramu	30940	54	M	J / DU	–	HP	+	–	Palliative	CI	Alive
38	Anandhavallli	31295	41	F	J / DU	+	CC		–	Palliative	Uneventful	Alive
40	Krishnamoorthy	35096	61	M	J / DU /WL	+	PC		–	Palliative	WI	Alive
41	Kanagaraj	40599	31	M	J / DU /WL	–	M		–	Palliative	Uneventful	Alive
42	Antony	43050	43	M	J / DU /WL	+	HP	+	Well Diff.	Whipple	Fistula	Alive
43	Manikandan	46504	55	M	J / DU /WL	–	HP	+	–	Palliative	Uneventful	Alive
44	Savithri	44303	32	F	J / DU	+	PC		–	Palliative	Uneventful	Alive
45	Rani	59303	45	F	J / DU /WL	+	HP	+	Mod. Diff.	Whipple	Uneventful	Alive
46	Stephenraj	58383	58	M	J / DU /WL	–	M		–	Palliative	CI	Alive
47	Sivalingam	58493	65	M	J / DU	–	HP	+	–	Palliative	WI	Alive
48	Muthu	58695	45	M	J / DU /WL	+	HP	+	–	Stenting	Uneventful	Alive
49	Revathy	60283	47	F	J / DU /WL	+	HP	+	–	Palliative	DGE	Alive
50	Munisamy	61394	67	M	J / DU /WL	–	HP	+	–	Palliative	Uneventful	Alive

LEGEND:

J - Jaundice
WL - Weight Loss
DU - Dark Urine
CCP - Chronic Calcific Pancreatitis

HP - Ca. Head of Pancreas
PC - Periampullary Carcinoma
CC - CholangioCarcinoma
CGB - Ca. Gall Bladder
M - Miscellaneous

WI - Wound Infection
CI - Cholangitis
DGE - Delayed Gastric Emptying